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Knowledge regarding
Localization in the
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BY

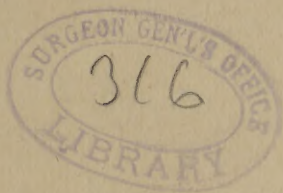
LANDON CARTER GRAY, M. D.,

PROFESSOR OF NERVOUS AND MENTAL DISEASE IN THE
NEW YORK POLYCLINIC.

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THE PRESENT STATE OF OUR KNOWLEDGE REGARDING
LOCALIZATION IN THE CORTEX CEREBRI.*

BY LANDON CARTER GRAY, M. D.,

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ALTHOUGH it has only been within a very recent time that cortical localization has been precise, the doctrine is yet an old one. The evidence of varied mental action, of which every people must have been conscious after the attainment of a certain grade of civilization, necessarily led the ancients to the conception of cerebral compartments. But these conceptions were confined to such vague ideas as those of the early Arabian physicians, who placed sensibility, intellect, judgment, and memory in the ventricles; or as those of Albertus Magnus, bishop of Ratisbon, who mapped out, on a brazen head of his own manufacture, the frontal region as the site of general sensibility and imagination, the vertex as that of intellect and judgment, the occiput as that of memory; or as those of Mondino di Luzzi, who in the fourteenth century thought each ventricle to be endowed

* Read before the Medical Society of the County of Kings, March 15, 1887.

with a particular form of intellectual force; or as Guy de Chauliac, surgeon to the Avignon popes, evolved a fanciful cerebral localization; or as Montagnanus, who in 1491 published a chart of the brain with regions indicated for the "sensus communis," for the imaginative cell, for the cogitative cell, for the memory cell, and for the rational cell; or as Ludovico Dolci, Thomas Willis, Swedenborg, Descartes, Vieussens, or Haller, who gave free rein to their imagination, unchecked by any foreshadowing of what the centuries might disclose. It was not, however, until the early part of the eighteenth century that the idea was clearly outlined. Haller and Zinn, in 1756, professed to have seen convulsive phenomena after injury of the cerebral white substance; but these observations were soon overlooked. It is too much the fashion at the present day to overlook the services which were then unconsciously rendered by Gall and Spurzheim. Both of these Germans were excellent cerebral anatomists, both have left works of unquestionable scientific merit, both were persecuted for opinion's sake,* and both lived in the thick of the times which bred Mesmer and Hahnemann, and the pseudo-scientific, semi-mystical mixture of truth and charlatanism of which Mesmer and Hahnemann were the most illustrious exponents. But Gall and Spurzheim were above the level of Mesmer and Hahnemann, as, apart from the quality of their other work, is evidenced by the fact that they counted among their believers such men as Broussais, Bouillaud, Andral, G. Comte, and, with certain qualifications, also Reil and Hufeland. Even the great Goethe thought their system of phrenology of sufficient importance to enter into an elaborate argument against it. But it is

* Gall was forbidden to lecture in Vienna, because, forsooth, it was feared that his views would disturb the minds of men in their feudal beliefs and feudal loyalty.

not surprising that this idle pretense of diagnosticating the mental faculties by the protuberances upon the external surface of the skull should have met with no enduring reception, or that, following the teachings of Magendie and Flourens, the medical profession should have veered to the other extreme of disbelief in any cortical localization whatsoever. It is, however, a remarkable illustration of the limitation of the human faculties that such an expert physiologist as Flourens should have failed to obtain any of the diversified and startling cortical phenomena which any tyro knows how to obtain to-day. Flourens, writing in 1824, and reiterating his assertions in 1842, stated unequivocally that removal of the brains of animals produced mental impairment in proportion to the amount of cerebral tissue removed, and not with any relation to the locality. Opposed to his teachings were the clinical observations of Bouillaud, who in 1825 recognized that loss of the memory of words which has in later days come to be known as aphasia; of Marc Dax, who in 1836 * located this symptom in the frontal lobe; of Broca, who in 1863 made a more precise localization within the third frontal convolution; of Andral, in 1834, who then reported cases of paralysis of the arm and leg from cortical disease; of Panizza, who in 1855 reported two autopsies which clearly indicated a relationship between sight and the parieto-occipital region of the hemisphere. It is noteworthy that, in the lively discussion of the subject of aphasia at the Parisian *Académie de médecine* in 1864, much skepticism was expressed, and even the gifted Trousseau, in spite of his peerless clinical instinct, strenuously gainsaid that clinical and anatomical precision of Broca's which time has magnificently vindicated. Notwithstanding these clinical revolts,

* Although he first published his views through his son, G. Dax, before the Academy in 1863.

the influence of Flourens was paramount with such physiologists as Longet, Magendie, Matteucci, Budge, and Schiff until 1870—nearly half a century. Yet the same old vague line of thought was still germinating. In 1867 Theodor Meynert began his brilliant and original series of articles upon the structure of the cerebrum, and announced his theory of the projection-system—*i. e.*, of a nerve-tract which should connect with the cortex of the hemisphere all sensory surfaces and the voluntary muscular system. Through this tract all sensations should travel inward, all motor impulses should travel outward. As he puts it epigrammatically: “A cross-section of the *crus cerebri* would therefore implicate the whole organism, which would simply be smell-less and blind,” inasmuch as the olfactory and optic nerves do not pass to the periphery through this channel. The motor portion of this projection-tract went, he maintained, through the nucleus caudatus and the nucleus lenticularis, to the frontal lobe, while the sensory went, by way of the optic thalamus and the corpora quadrigemina, to the occipital and temporal lobes. The epoch-making experiments of Fritsch and Hitzig, in 1870, lent a remarkable confirmation to these teachings, at the same time that they revolutionized the existing ideas of cortical physiology. These original observers demonstrated, in direct contradiction of all previous experimenters, three important series of facts:

I. That a portion of the convexity of the cerebrum is motor in its function, while another portion is not motor.

II. The motor portion, speaking generally, lies more anteriorly, the non-motor portion lying more posteriorly.

III. Electrical irritation of the motor portion gives rise to combined muscular contractions of the opposite side of the body.

They mapped out in the brain of the dog the centers for the neck muscles (Fig. 1), the extensors and adductors

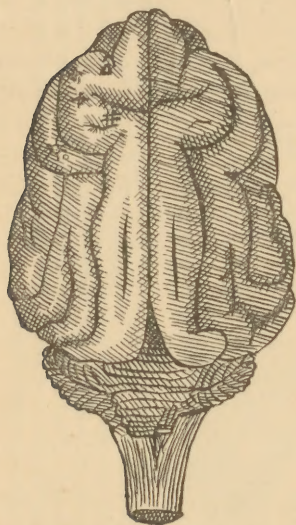


FIG. 1.—(After Fritsch and Hitzig.)
 Δ, neck muscles; + (anteriorly),
 extensors and adductors of fore-
 leg; + (posteriorly), flexors and
 rotators of fore-leg; # hind-leg;
 ⌘ facial.

of the fore-leg (+ anteriorly), the flexors and rotators of the fore-leg (+ posteriorly), the muscles of the hind-leg (#), and the facial muscles (⌘). They also removed with a scalpel the center of the fore-leg, and found that the dog in walking set this foot down clumsily, seemingly without intention, sometimes to one side, sometimes to the other, and that this leg slid outward. In standing, the same phenomena appeared, and it was also seen that the foot was occasionally set down upon the dorsal surface. In sitting upon the hinder parts, both fore-feet resting upon the ground, the affected fore-leg gradually slid outward,

until the animal lay prone upon the corresponding side of the body.

The paper of Fritsch and Hitzig gave birth to an enormous literature, to which addition is being made daily. The subject has been furiously discussed, and, in one instance, almost led to a personal altercation between two distinguished physiologists. Confirmation of the doctrine thus advanced by Fritsch and Hitzig, of circumscribed cortical centers, came from every side. It was, however, soon

shown that the paralysis resulting from destruction of such a center might entirely disappear, although the destruction

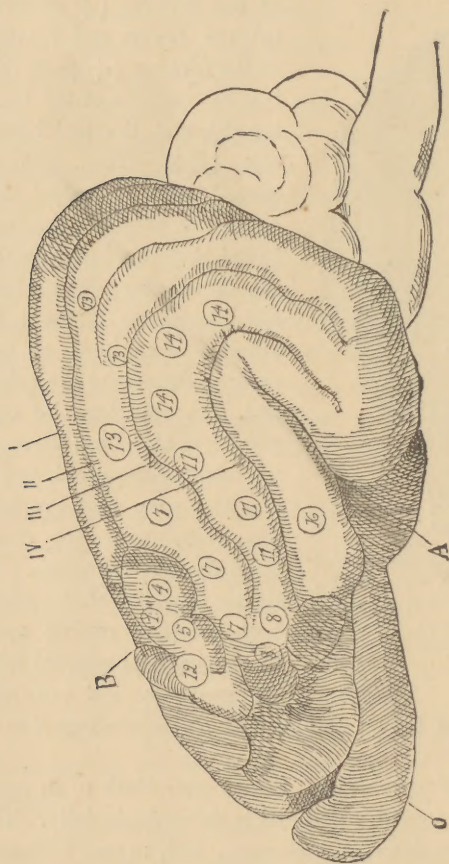


Fig. 2.—(Ferrier.) 1, opposite hind-leg advanced; 2, lateral movement of tail; 3, retraction and adduction of fore-leg; 4, closure of eye through orbicularis and zygomatics; 5, elevation of shoulder and forward extension of fore-leg; 6, closure of eye through orbicularis and zygomatics; 7, opening of mouth and tongue movements; 8, retraction and elevation of mouth angle; 9, opening of mouth and tongue movements; 10, retraction of mouth angle; 11, opening of eyes, pupillary dilatation, movement of head and eyeballs to opposite side; 12, movement of eyeballs to opposite side, with slight diagonal deviation; 13, pricking or sudden retraction of ear; 14, torsion of nostril. The figures omitted indicate centers inert in the dog, though active in monkeys, according to Ferrier.

of the center was proved by an autopsy to be complete. This seemed, at first sight, to be a serious objection to the theory; but further experimentation on the monkey tribe,

as well as the results of disease in the human being, demonstrated that this recovery from the resultant paralysis of destruction of a cortical center was only observed in those

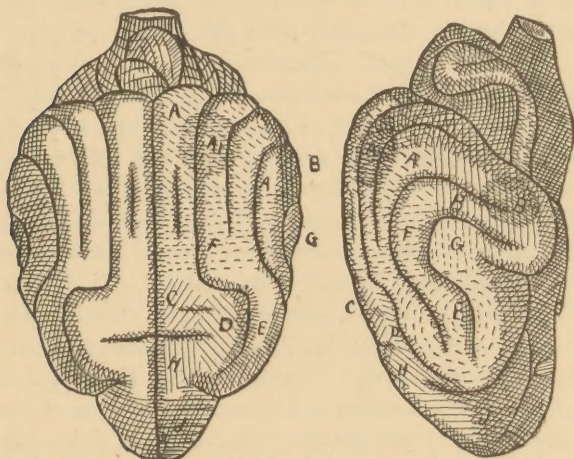


FIG. 3.—(Munk.) A, sight sphere; B, auditory sphere; C-J, sensory sphere; D, fore-leg center; C, hind-leg center; E, head center; F, eye center (for muscles of the eye); G, ear center (for muscles of the ear); H, center for neck muscles; J, center for trunk muscles.

animals in which the cerebrum played a subordinate part. Some lower animals, for example, run and walk soon after birth. In these, removal of the hemispheres has only a temporary effect, because in them the masses of gray matter lying beneath the hemispheres are relatively of larger development, and perform those functions which are relegated higher up in the scale of nervous evolution to the hemispheres. But even those who have accepted Fritsch and Hitzig's declaration of circumscribed motor centers have not been in perfect accord with them in regard to the exact localization of each area. Ferrier, of London, has been greatly at variance with them, as will be seen by a

comparison of the accompanying figure with Fig. 1. And others, while recognizing the existence of cortical centers of which electrization gives rise to muscular movements, and of which removal causes paralysis, have interpreted the phenomena differently from Fritsch and Hitzig. By one of those singular coincidences which have occurred more than once in the history of medicine—showing how public strains of thought will occasionally lead to precisely similar deductions in widely separated and differently environed individuals—Goltz in Strassburg, Munk in Berlin, Moeli in Berlin, and Tripier in Lyons, demonstrated that limbs paralyzed by removal of their cortical centers showed a loss of sensation as well as of motion. To the excitable area of the cortex, therefore, which Hitzig had regarded as purely motor, Munk gave the name of “sensory sphere” (*Fühlssphäre*). (See Fig. 3.) But Munk carried his experiments still further, and showed that lesions of the occipital lobe produced peculiar disturbances of sight. If the area A (Fig. 3) were removed on both sides from the two hemispheres of a dog’s brain, he would see things, would avoid objects placed in his path, but would be unable to recognize these things and objects. He would view with indifference his master, other dogs, his food; would not wink at the approach of a light or a finger to the eye; would not recognize the whip, at sight of which he had been taught to go into the corner. But if he were permitted to take cognizance of his master, his food, etc., with the other senses, he recognized them as usual—thus, he ate food after smelling it, or retreated before the whip when it was cracked. This non-recognition of objects seen was termed by Munk “soul-blindness” (*Seelenblindheit*). A better name, I think, would be “mental blindness.” The removal of this same area A on one side produced mental blindness of the opposite eye. On the other hand, removal of the area A, which surrounds

A, caused absolute blindness;* and Munk professes to have determined that different sections of the retina are connected with different portions of this area A. He asserts that removal of the inner medial half of this area produces absolute blindness of the inner half of the opposite retina; that removal of the inner third of this area produces absolute blindness of about one third of the inner part of the opposite retina; that the anterior half bears the same relation to the upper half of the opposite retina, while the posterior half is connected with the opposite lower retinal half. But the most curious of all that Munk professes to have demonstrated in this connection is that the fibers from the *macula lutea*, the retinal point of most distinct sight, terminate in the area A, so that the symptoms of mental blindness are associated with absolute blindness as regards distinct sight. Upon this anatomical peculiarity Munk bases an explanation of the mental blindness. Distinct sight, he says, through the fibers of the *macula lutea*, gives us usually our visual impressions. When the cortical termination of these macula-luteal fibers is removed, a certain time must elapse before we can become accustomed to receiving visual impressions through fibers from other parts of the retina, which, as we have seen, terminate in different parts of the area A; and, as a matter of fact, mental blindness is a symptom of short duration. It may be stated here at once, though, in anticipation of what should follow, that, however well this explanation may apply to dogs, it does not hold true of man, for cases have since been reported of mental

* This absolute blindness Munk would have us call "cortical blindness" (*Rindenblindheit*)—a perfectly meaningless and pedantic term, it seems to me, that can only serve the purpose of making the confusion of cerebral nomenclature worse confounded. Munk himself has felt obliged to attach a parenthetical explanation almost every time he has used it.

blindness without accompanying impairment of distinct sight, and, contrariwise, of lesion of the *macula lutea* without mental blindness. Munk also indicated the area B of the parietal lobe as the center of hearing, removal of it giving rise to mental deafness of the same nature as the mental blindness just described.

The nicety of observation, the judicial tone, the care, patience, and time evidenced by each successive communication—all combined to attract great attention to the dicta of this Berlin physiologist. Nevertheless, although he soon had many followers, he did not meet with universal confirmation. Ferrier, of London, whose experiments began in 1875, following those of Fritsch and Hitzig, and supplementing these in many matters of detail, flatly contradicted Munk in regard to the sensory nature of the excitable region, as well as concerning the optic and auditory centers and their nature.

It will be readily seen that at this stage of its evolution the subject was involved in the most inextricable confusion. It became simply a matter of bias as to what a man should believe. Each physiologist maintained that there were inherent defects in the method of experimentation of the others, and each brought forward to the support of his own views facts that had either to be denied *in toto* or else accepted, to add to the doubt (compare Figs. 1, 2, 3, 4). It is not to be wondered at, then, that Goltz, of Strassburg, afterward supplemented by his pupil, Loeb, should have voiced the sentiment of many by denying altogether the doctrine of circumscribed cortical centers. He repeated especially the experiments of Munk, and stated that he obtained entirely dissimilar results. But it may be said here, once for all, that, although Professor Goltz has been extremely useful as a censor, his experiments do not warrant his conclusions; indeed, he refutes himself, so that, in simi-

lar terms to those which Shakespeare puts into the mouth of Hamlet addressing Rosenkrantz and Guildenstern, he has fretted the advocates of cortical localization, but he has not played upon them—he has not stopped their way.

But the continuance of the study upon human beings has been rewarded by more permanent conclusions. Pathology has gone hand in hand with physiology. Each has its advantages. The skull of a dog or an ape can be trephined at will, and just as much removed of the cerebrum as the operator desires; the animal can be kept constantly under observation, and often, when there are no fatal results, for a considerable length of time, and the operation can be repeated. Focal alterations of the human brain are rarely so localized, and can not, of course, be produced at will, or kept so well under observation; so that conclusions which may rest upon a few months of physiological experiment can not be contradicted or verified except by years of widespread and isolated observations upon the human subject. On the other hand, human beings, with their superior intelligence, are infinitely better subjects for testing the manifold details of motion and sensation. It is, therefore, I think, the better proof of the truth of the doctrine of localization that the experiments of physiologists upon the dog and monkey tribe should have tallied so well with the experiments of disease upon the human being. The individual facts of the latter kind, upon which my conclusions are based, are too numerous to be considered in a work of this kind. Any one who desires to review the testimony will find full references in the appended bibliography. It must suffice my purpose to indicate the conclusions.

Regarding the human brain, there are two sets of facts—one set that is indisputable, another that is still under discussion. Let us first consider the former.

Look at Fig. 4, in which the different convolutions are

indicated. The ascending frontal and parietal convolutions are divided into three equal parts. Of these, the upper third, with the adjacent portion of the base of the first and second frontal convolutions, contains the center for the lower extremities; the middle third, with the adjacent base of the second frontal convolution, contains the center for

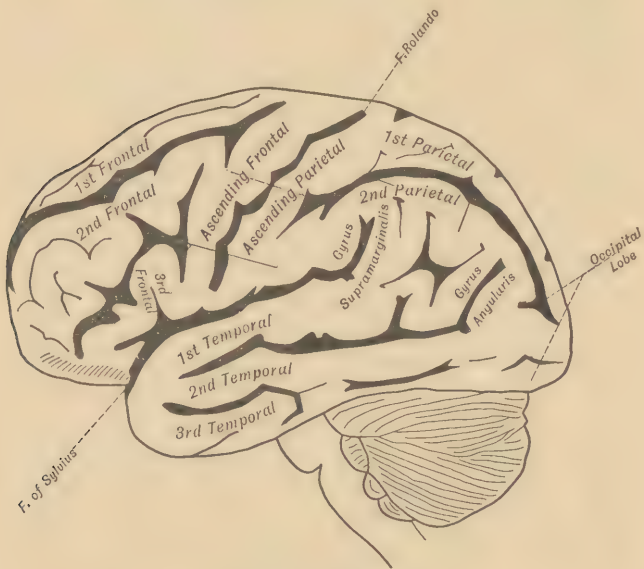


FIG. 4.—(After Ecker.)

the upper extremities; the lower third, with the third frontal convolution, contains the centers for the face, neck, and speech muscles. Fig. 5 represents the medial surface of the hemispheres. The so-called "paracentral lobule" is the medial surface of the upper part of the ascending parietal and frontal convolutions, and therefore contains the center for the lower extremities, although lesions of this medial

surface are comparatively rare. It will be perceived that this paracentral lobule is the only part of the motor convolutions that reaches to the medial surface of the hemispheres. This area upon the convex and medial surfaces is that of what may be called the "facio-phonetic-skeletal region." Lesions of it produce impairment, in the manner indicated, of the upper and lower extremities, of the facial and neck muscles, and of the motor mechanism of speech. It may justly be regarded as proved that the paralysis of the limbs and the face is both motor and sensory, that the motor and sensory paralysis are not always of equal intensity, that the one may occur without the other, and that the area within which sensory paralysis may be produced is of larger extent than the motor area, inasmuch as the former embraces the

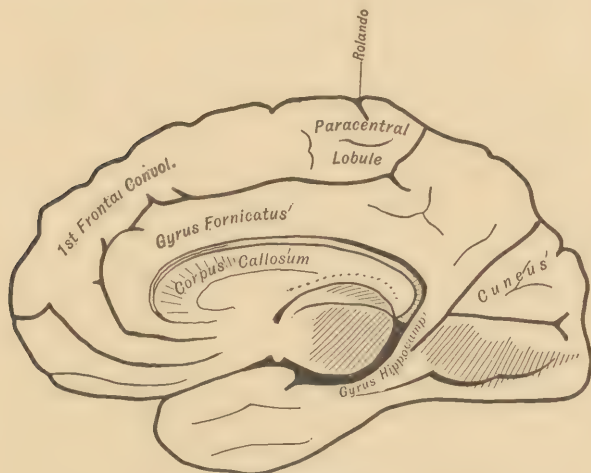


FIG. 5.—(After Ecker.)

two parietal convolutions (Fig. 4). It will be seen that I have mapped out these areas somewhat indefinitely.

In matters of this kind one is greatly tempted to draw precise circles for each center, and, doubtless, positivism of this kind saves much trouble to those of great faith; but I can not reconcile facts to such a sharp delimitation. In truth, the areas overlap one another, just as the convolutions pass imperceptibly into one another, and the time will never come when a man will be able to mark a line on a convolution and say that it is a precise boundary-line between two centers, so that at one one-hundredth part of an inch to one side there will be certain symptoms, and totally different ones at one one-hundredth part of an inch to the other side. The centers can only be *approximately* demarkated, not *absolutely*.

There are also good clinical reasons for believing that each different kind of sensation—the tactile, pain, temperature, and muscular senses—has a cortical center of its own; but it has as yet only been determined that the muscular sense has probably its center in the parietal convolutions.

The center of sight is to be found in the occipital lobe and the angular gyrus (Fig. 4). There has been a fierce discussion regarding this center between the followers of Munk and those of Ferrier, the former denying that the angular gyrus had any part in this center, the latter affirming that any visual impairment must implicate both angular gyrus and occipital lobe. But the experiments of Luciani and Seppilli, and the cases collected by these gentlemen, warrant the assertion that the center embraces both angular gyrus and occipital lobe, although with this distinction, that lesion of the angular gyrus alone produces mental blindness, while lesion of the occipital lobe produces absolute blindness of the same half of the two retinae (lateral *hemipopia* *). Brill has reported a case of color-blindness in

* Hemipopia is derived from the two Greek words “*ἥμις*, half,” and “*ᾠπτομαι*, to see,” meaning therefore half sight. There is another

which the lesion was in the cuneus, the medial portion of the occipital lobe; and Seguin and Hun have reported cases of hemiopia due to disease of the cuneus and the adjacent temporal convolution. It is not yet certain whether disease of the angular gyrus causes visual symptoms by implication of the optic tract, which passes just beneath it, or whether the angular gyrus is itself a true terminal center of some fibers of the optic tract.

The center of hearing may be located in the first and second temporal convolutions (Fig. 4), although this area does not seem to be so constant a center as some others in the cortex, for the writer and Kussmaul, perhaps also Westphal, have reported cases in which a lesion was located here without the expected auditory symptoms. It is curious, however, that the left lobe seems to be mainly affected, the right side seeming to be of greatly subsidiary importance, Luciani and Seppilli stating that it is never affected alone, while the lesion is very seldom in both temporal lobes. It is curious, too, that the cases have so far always presented the symptom of mental deafness analogous to the mental blindness, as described above, and never any absolute deafness.

The cases of cortical production of loss of smell or loss of taste have been too scanty to define the centers of those two special senses, although it is probable that the olfactory center is in the hippocampal convolution (Fig. 5).

somewhat similar word, which has led to much confusion—hemianopsia, from “*ἡμιος*, half, *ἕν* (for *ἕνα*), each, and *ὄψις*, sight,” meaning also half sight. It seems to be agreed at the present day that by arbitrary custom hemiopia shall mean the condition of the retina, while hemianopsia shall be applied to the crossing of the rays of light in the media in front of the retina. Thus, a left *hemioopia* will indicate that the left halves of both retinae are blind, so that the patient, not being able to see objects to the right of either eye, shall be said to have a right *hemianopsia*.

As Goltz has combated the physiological advocates of cortical localization, so has the distinguished Brown-Séquard vigorously opposed the clinical defenders of the same doctrine. But Brown-Séquard's collection of cases is open to two fatal objections: Firstly, his cases antedate the pe-

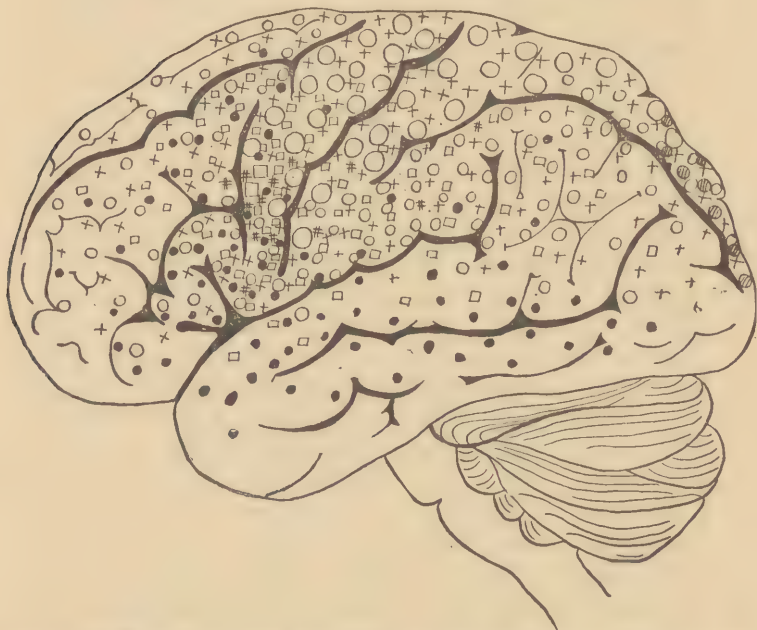


FIG. 6.—(After Exner.) ○, upper extremity; +, lower extremity; □, facial; #, hypoglossus; ●, speech; ●, sight.

riod of accurate knowledge of cerebral topography; secondly, he fails to take into account the fact, which had been demonstrated by the embryological researches of Flechsig, that the motor tracts do not decussate or pass over to the opposite side in all human beings, and that consequently a

paralysis may be on the same side as a cerebral lesion, and yet not militate against the doctrine of localization.

Let us now consider the facts that are still under discussion.

In some cases a lesion outside of what has been deemed the center will produce the symptoms which result from lesion of the center itself; and, again, lesions of the center that are ridiculously minute will produce as marked symptoms as if the whole center were implicated. For these reasons Exner constructed the accompanying chart (Fig. 6), in which it will be perceived that lesions at divers points of the cortex have produced the same symptoms, although there is for each function a certain area within which the lesions are mainly grouped. This area of densest grouping of lesions Exner regards as the true center. The figures which represent the experiments on dogs and monkeys of Luciani and Seppilli tally remarkably with the pathological data collected by Exner, as will be seen by Fig. 7. What, then, is the meaning of the production of similar symptoms by lesions outside of the center? I take it to be that lesions at a distance from the true center can inhibit the action of that center. These phenomena of inhibition are very familiar to us in the peripheral nervous system. All practitioners are aware of the many nervous disturbances which are removed by removal of a tight and redundant prepuce, a urethral stricture, a diseased ovary, a post-nasal catarrh, errors of ocular refraction, tumors of peripheral nerves, etc. In these instances the nerve tract to the spinal cord or the cerebrum is a comparatively long one. But in the cerebrum the commissural tracts, connecting different areas of the cortex, are far shorter and more numerous than those leading from the nervous axes to the periphery. How much more effective, then, must be the inter-cortical inhibition. But this very probability also makes evident—what has

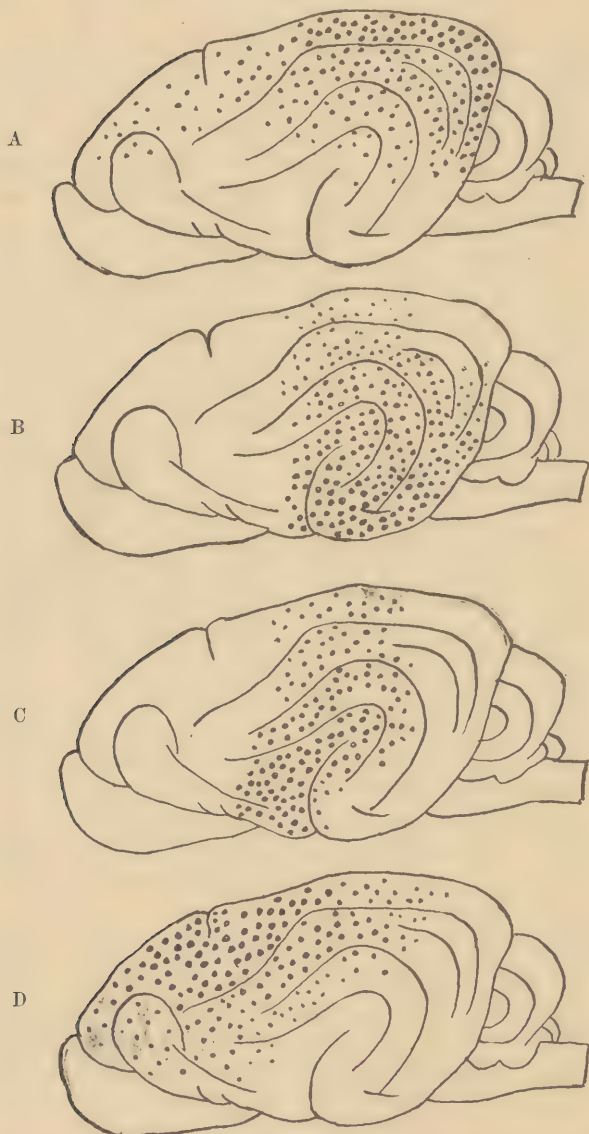


FIG. 7.—(After Luciani and Seppilli.) A, center of light ; B, center of hearing ; C, center of smell ; D, sensory sphere.

been singularly disregarded by writers upon localization—that the locality of a lesion can only be diagnosticated with *probability*, not with *certainty*.

From this review we perceive that the doctrine of cortical localization is far too well grounded upon facts of eternal verity to be flippantly sneered at, although much remains to be done in the way that has been hewn out of primeval ignorance and acquired obstinacy. Like all truths that have lurked undiscovered for centuries, except those that do not require skilled experimentation or trained observation, it has had to rely upon the testimony of a cloud of witnesses, each one varying in competency or bias, and the result has been the ordinary one of a long trial of issues of fact before an ordinary jury—a failure to convince every one. But the jurors of science can wait for all time, the trial is never closed, and no verdict, however conclusive it may seem at the time it is given, will stand for one hour in the face of a newly discovered fact. In spite, therefore, of uncertainties about minor points of detail, we must admit that we have localized the cortical centers for the motor and sensory nerves of the limbs and face, for the mechanism of speech, for the optic nerves, and probably also for the auditory nerves.

The question now arises, What is the meaning of these centers in the cortex? It is simply that each center is the cortical area in which certain nerve-tracts terminate. Mind is, to a certain extent, a mystery, and will probably remain so, to a certain extent, for many generations, perhaps ages, yet to come. But this mystery called mind is dependent for its healthy manifestation upon the structural integrity of the cortex, and of the whole of the cortex. Hence the cortex is justly called the organ of the mind. This mind can only come into communication with the world that lies outside of the skull-cap by the material highways of the

nerves, some of which carry impressions into it, others of which carry actions out from it. Thus, mind sees by means of the optic nerves, hears by means of the auditory nerves, tastes by means of the gustatory, feels by the tactile, muscular, pain-bearing, and temperature-telling nerves, acts by means of the motor nerves, and educates itself by means of them all; and the so-called "centers" are simply the areas of the cortex in which these different nerve-tracts terminate. These centers are the cortical stations for the great trunk-lines of the peripheral nerves. It is easy to understand that there may be regions of the cortex in which there are none of these terminal nerve-stations, none of these centers, and that lesions of these regions may therefore not produce any impairment of the peripheral nerves of sense and motion, but rather give rise to purely mental symptoms, disconnected with motion or sensation. It is also easy to understand that, as has been indicated, lesions of the cortex outside of the centers should impair the action of the center itself by inhibition along commissural nerve-fibers connecting one cortical area with another. Nor is it difficult to unravel the seeming complexity of certain symptom-groups which have puzzled clinicians until a recent date. For example, aphasia may be both motor and sensory. When the lesion is seated in the third frontal convolution (Fig. 4), the aphasia is motor, the patient's mind is unable to act upon the muscular machinery of speech, and he can not give expression to words. When the lesion is in the first and second temporal convolutions, the patient loses the memory of words, fails to recognize spoken words; he has the so-called "word-deafness," although he may hear perfectly well, and although he may be able to articulate words well enough. If both the third frontal convolution and the two temporal are diseased, the patient can necessarily neither articulate nor recollect words. If a lesion implicates the an-

gular gyrus, he may fail to recognize objects that he sees, or, in some instances, the non-recognition of objects seen may be confined to words; and if the lesion extends into the occipital lobe, this non-recognition of objects seen may be complicated with blindness of one half of the retinae (*hemioopia*). Mind itself, in all these symptoms, may be intact, left isolated, as it were, in the cortex, shut off from its motor and sensory communication with the outer world.

Note.—The writer desires it to be understood that there are many facts pointing to more localization than has been indicated in the foregoing article, and some of them seemingly quite precise too. For instance, Dr. A. Fraenkel describes (*Charité-Annalen*, 1886) a case observed in the Charité Hospital of Berlin in which, during life, a diagnosis was made of meningitis because of the retraction of the neck, and in which there was found post mortem a softening of the base of the second frontal convolution extending into the middle third of the ascending frontal (see Fig. 4), thus confirming in a remarkable manner the localization of the center for the neck muscles made by Wernicke several years ago, after careful consideration of the experiments upon apes and monkeys (see Fig. 1, Δ , and Fig. 3, II). But again and again has experience taught us, in the seventeen years that have elapsed since the original discovery of Fritsch and Hitzig, that it is not safe to locate a center upon one or two observations. The writer has, therefore, only spoken of those localizations which he believes to have been adequately settled.

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